

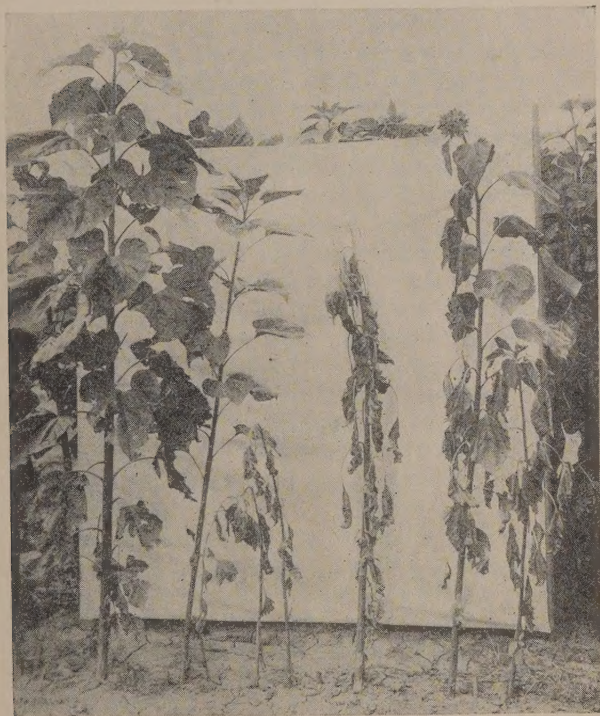
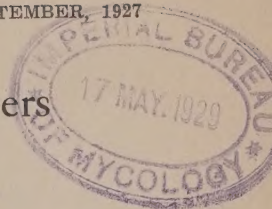
Sclerotinia Wilt of Sunflowers

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WILTED AND NORMAL SUNFLOWERS

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Sclerotinia Wilt of Sunflowers¹

The first specimens of wilted sunflowers received by the Plant Pathology Department were collected in August, 1920, at Chinook, Montana, where the disease was causing considerable loss. Wilted sunflowers were found in the sunflower plots on the Experiment Station Farm, at Bozeman, and in commercial fields in the Bitter Root Valley the same year. Thus sunflower wilt was observed in three widely separated localities in Montana at about the same time. Since sunflower silage was apparently becoming popular, investigation of this destructive disease was begun as an Experiment Station project.

Most of the inquiries concerning sunflowers in the United States before 1900 were in regard to their cultivation, the use of the seeds for human and animal food, and the manufacture of oil (Wiley, 33).² The wild sunflower (*Helianthus annuus* L.), from which the cultivated variety was developed, is native in the Great Plains region of North America from Nebraska to northern Mexico. It was first cultivated in Spain about the middle of the sixteenth century. This was soon after the Spanish expeditions to Peru and the Santa Fe region of New Mexico. Dr. Asa Gray (13), who carefully studied the history of the sunflower, expressed the opinion that the wide heads distinguishing cultivated from wild sunflowers developed before its introduction into Europe.

The first report on the use of the whole sunflower plant for silage was in the Maine Agricultural Experiment Station Report of 1895. From 1895 to 1915 very little work was done on the use of sunflowers for this purpose (Vass, 32). In Montana the first work on sunflowers for silage was begun by the Experiment Station in 1915. Mammoth Russian sunflowers were used in the first experiments. Since 1915 valuable data applicable to Montana have been secured regarding varieties, methods and dates of planting, seed requirements, adaptability to dry land, yields, quality, and uses for silage. This work was

¹The writers are indebted to Professor D. B. Swingle for many helpful suggestions.

²Figures in parentheses refer to Literature Cited, page 29.

published in bulletins by Arnett, Atkinson, Blish, Jensen, Joseph, Nelson, and Tretsven (1, 6, 7, 15, and 17).

For silage, diseased plants which wilt early in the season represent a complete loss, while the value of plants which wilt only a few days before cutting is only slightly impaired. In general, diseased plants that wilt and droop more than ten days before harvesting are practically worthless because the stems become dry and hard, and the leaves are broken into small fragments in harvesting. Under average conditions a loss of 0.1 to 10 per cent is a conservative estimate in commercial fields where rotation is practiced.

Various authors (22) have reported sunflower wilt in Idaho, Washington, Oregon, Michigan, British Columbia, Manitoba, Ontario, Quebec, and Prince Edward Island. Swingle (30) and Morris and Swingle (21) described sunflower wilt in Montana. Henry and Gilbert (14) described the disease in Minnesota. Jones (16) reviewed the literature dealing with the occurrence of *Sclerotinia* in sunflowers in Europe.

Sclerotinia wilt of sunflowers has not been reported from the sections of the United States producing sunflower seed, such as southeastern Missouri, southern Illinois, and the San Joaquin Valley of California. Much of the seed used for commercial planting is imported from Russia where the raising of sunflowers is an important industry. Autokolskaya (2) reported *Sclerotinia libertiana* as causing wilt of sunflowers in Russia.

SYMPTOMS AND SIGNS

Cultivated sunflowers (*Helianthus annuus* L.) are attacked by *Sclerotinia libertiana* Fuckel during most of the growing season, the fungus usually entering through the roots. The disease usually appears in the Gallatin Valley about July 1st. The first symptoms are wilting of the leaves of affected stems, and the appearance of prominent cankers on the crowns and bases of the stems (fig. 1). These cankers have a rather soft consistency, a greenish-gray to brown color, and a water-soaked appearance. The cankers enlarge rapidly and affect the lower 8 to 30 cm. of the stems. Dense, white mycelium (mold) appears on the cankers and produces fuzzy, white bunches that soon become irregular, hard, black sclerotia (overwintering

bodies) 3 to 10 mm. in diameter. Water leaking from the bases of cankers sometimes wets the soil for about 2 cm. around diseased crowns while the adjacent surface soil is dry. In shaded spots white mycelium occasionally grows out along the surface of the soil for 2 cm. around cankered stems.

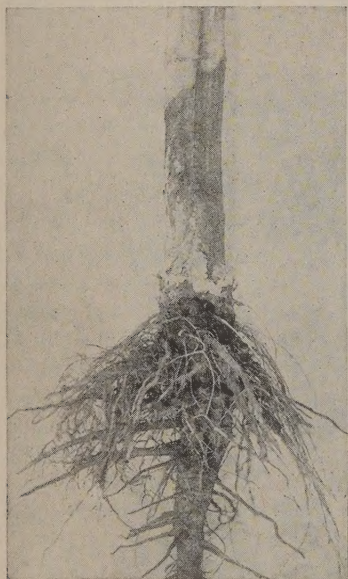


Fig. 1.—Base of a sunflower stem showing a canker and the white mycelium (mold) of *Sclerotinia*.

The crown cankers frequently are quite large before any of the leaves begin to wilt, while in other cases the lower leaves wilt before the crown cankers appear. Sunflower roots are badly diseased before the crown cankers appear, and diseased plants are easily pulled up because most of the branch roots have decayed. Flat sclerotia 0.3 to 3 cm. long commonly occur on the largest roots, and smaller sclerotia are found enclosing some branch roots (fig. 2 and Pl. I, H, L, M, N). The leaves of diseased sunflowers soon wilt completely and the heads then droop (see title page).

Sclerotinia often decomposes the parenchymatous tissues in the cankered parts of stems and these stems frequently break over in the cankers. The lignified bast and vascular bundles

in these cankers separate and give them a shredded appearance. The rupturing discloses many sclerotia produced by the fungus in cavities left by the decomposition of the pith (figs. 3, 4). These sclerotia are black, with white mycelium adhering to parts of their surfaces, and are 1 to 118 mm. long (Pl. I, A. B). Lawrence (18), Baribeau (3), Bisby (5), Swingle (30), and Morris and Swingle (21) described sclerotia in sunflower stems. Massee (19) illustrates sclerotia in a *Chrysanthemum* stem. Cook (10) says that sclerotia of *Sclerotinia* occur in the central

cavities of dahlia stems. Poole (23) illustrates *Sclerotinia* causing a stem rot of large celery plants. Cankers sometimes occur in sunflower stems a meter above the ground and the stems break over in such cankers; this phenomenon was often seen in inoculated stems. About half of the diseased plants do not break over, and these dark brown stalks with their light brown, moldy, cankered bases present a characteristic appearance long after they have died.



Fig. 2.—Sclerotia (overwintering bodies) of *Sclerotinia* taken from the roots of Mammoth Russian sunflowers. $\times 1$.

When seasonal conditions permit, the fungus attacks the sunflower heads and partly or entirely rots them so that the remains crumble and fall to the ground; the cankers in the heads resemble those in the stems (fig. 6). Large, black sclerotia develop below the seed layers, while others form around the seeds and consist of black crusts containing many diamond-shaped holes through which the ends of the seeds project (Pl. I,

C-G and I-K). The seed coats of the seeds in rotted heads may be normal in size, depending upon the time of infection. However, they are often discolored and empty or contain only dwarfed embryos. Small sclerotia are often attached to seed coats produced in moldy heads (fig. 7). Sclerotia may also occur inside the seed coats (Pl. III, D); many were produced inside the seed coats of White Beauty sunflower seeds in 1927.

THE CAUSAL FUNGUS

Cultural studies of the fungus isolated from diseased sunflower tissues clearly showed that the fungus is *Sclerotinia libertiana* Fuckel. Jones (16) also showed that this fungus causes sunflower wilt.

Rabenhorst (24) gives *Sclerotinia sclerotiorum* Schroet. as a synonym of *S. libertiana*. Probably Schroeter was the first to use this combination. If so, the name, *S. sclerotiorum* (Lib.) Masee, is incorrect on the basis of priority. Since confusion exists regarding the name of this fungus, the old, commonly used name, *Sclerotinia libertiana* Fuckel, is used here. Lawrence (18) did not prove the genetic relationship between the conidial and ascigerous stages which he described for *Sclerotinia perplexa*, and the ascigerous stage of this fungus is not different from *S. libertiana*, so the name, *Sclerotinia perplexa*, appears to be invalid. The fungus causing the sunflower disease which he described was probably *S. libertiana*.

In the experimental plot and in a field at Bozeman in 1925, *S. libertiana* produced all the disease symptoms and all the different forms of sclerotia described for *S. perplexa*. A brownish-gray species of *Botrytis* was abundantly associated with *Sclerotinia* on the rotting sunflower heads. Many cultures of the two fungi were made under similar conditions and no similarities or genetic relationships were observed.

Sclerotinia libertiana from sunflowers grew very slowly at 0° to 5°C. and did not grow at 37°C. on corn meal, beef, sunflower, and potato agars. It grew slowly at 10°C. and rapidly at 15 to 25°C. Growth was very slow on beef agar. Abundant spherical to oblong-flattened sclerotia 2 to 10 mm. in longest diameter occurred in culture. Boyle (8), Bisby (5), and Stevens and Hall (28) say that gonidia occur in culture.

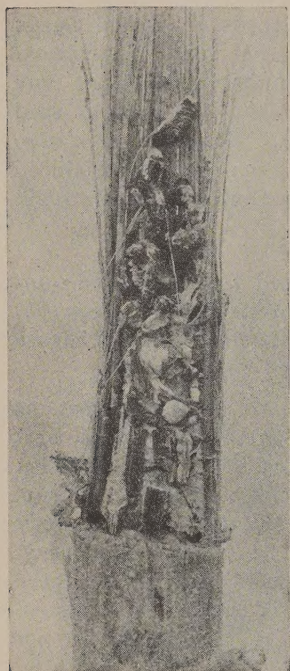


Fig. 3.—Sclerotinia canker in a sunflower stem showing sclerotia in the pith region. Shredded, lignified tissues remain in the old part of the canker.

The sclerotia which occur on and in stems and roots were described previously. Besides these, the sclerotia which *Sclerotinia* produced in rotting sunflower heads are particularly interesting. Solid, black sclerotia that sometimes enclosed old vascular bundles were abundant in the bases of rotting heads below the seed layers; they were 2 to 87 mm. long and often bore white mycelium on their surfaces (Pl. I, D-G). Another type of sclerotium occurred on the receptacle just below the seeds; these flat sclerotia were thin and had mycelium attached to their surfaces (Pl. I, C). One of these sclerotia was produced by a sunflower head in the field in 1927.

In the sunflower plot in 1925, 113 White Beauty sunflower heads high above the soil were rotted by *Sclerotinia*, and 54 of these bore sclerotial crusts in the seed layers. Many Mammoth Russian sunflower heads in an adjacent field were rotted and also bore sclerotial crusts in their seed layers. *Sclerotinia* usually entered through the margins of the heads and spread as a soft rot until the heads were partly or entirely decayed (fig. 6). Large areas of the seed layers were covered with dense mats of white mycelium which became large, flat sclerotia surrounding the seeds; hence, these sclerotia contained many diamond-shaped holes through which the ends of the seeds projected (Pl. I, I-K). Naturally infected heads in the field in 1927 produced sclerotial crusts. Lawrence (18) illustrated sclerotia of this type. These irregular sclerotial crusts were usually about 3 to 7 by 7 to 13 by 0.5 cm. Two exceptionally

large sclerotia were 12 by 20 by 0.5 cm. and 7 by 18 by 0.5 cm. Smith (27) says that *Sclerotinia* produced thin sclerotial crusts on lettuce. Sclerotia weighing 66 g. were collected from one sunflower head; these weighed only 37 g. after lying in the laboratory for six weeks. Sclerotia weighing 46 g. were collected from another head. Stevens and Hall (28) give the weights of sclerotia before and after drying.

Very small sclerotia occurred in the seed layers attached to the seeds and chaff; they were often flat and 1 to 2 mm. wide, and many of them grew in the interstices between the seeds. One case was seen in which a sclerotium occurred inside a seed coat in the place of the embryo; it had the shape of a seed (Pl. III, D).

Large sclerotia which had been kept in the laboratory for three or four months were planted in autoclaved, sandy soil. The surfaces of the soil were covered with wet cloth and the pots were kept in the greenhouse in a place exposed to sunlight; the soil was kept rather wet. Apothecia appeared in eight of the pots within six to ten weeks after planting. Nearly all of the apothecia were produced by sclerotial crusts from rotting heads (fig. 5). One large fragment of a crust sclerotium bore 125 stipes of which 45 had expanded apothecia; another fragment of a crust sclerotium in a different pot produced 44 apothecia; a few fragments of sclerotial crusts in another pot bore 176 apothecia; two pots containing sclerotia from sunflower roots produced 14 apothecia; one sclerotium from the interior of the base of a sunflower head bore 2 apothecia; and a sclerotium from the pith cavity of a stem produced 1 apothecium.

Sclerotia which had overwintered in the field inside a sunflower stalk shredded by *Sclerotinia* were brought into the greenhouse and planted in a pot of sand on May 13, 1926; 10 large apothecia appeared within the next six weeks. On September 15, 1926, while digging potatoes in a plot which produced wilted sunflowers in 1925, small numbers of apothecia of *Sclerotinia libertiana* were collected under three cloth potato plant cages. The stipes of the apothecia were traced down to sclerotia. These sclerotia had remained dormant in the soil since the fall of 1925, and finally produced apothecia in the damp soil under the potato plant cages.

On September 1, 1927, at least 1000 apothecia of *Sclerotinia libertiana* were found widely distributed in a 6-acre field of Mammoth Russian sunflowers on the Experiment Station farm. These apothecia were attached to stem and root types of sclerotia left in the soil by wilted sunflowers in the preceding year's crop. This field produced sunflowers for three consecutive years, with a rapid increase in the percentage of wilt each year. Their presence was probably due to the unusually wet fall weather. Production of ascospores so late in the season is of little value to this fungus, for large sunflowers are resistant to infection and are soon cut for silage.

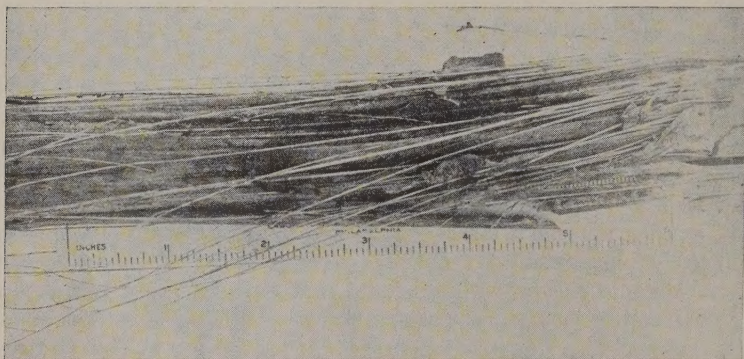


Fig. 4.—A sunflower stem severely affected by *Sclerotinia*, showing the prominent shredding caused by the decomposition of the parenchymatous tissues. Most of the fibers left are vascular bundles.

Hundreds of measurements of apothecia, asci, paraphyses, and spores were made from apothecia secured from the greenhouse pots and the field. Mature apothecia were usually 4 to 15 mm. wide, and avellaneous brown according to Ridgway's color chart. Apothecia produced in the field were larger than those in the greenhouse. The dimensions of the asci were 83 to 140 by 6 to 11 microns (averages about 100 to 130 by 6 to 9 microns). Blue tips were visible when asci were treated with iodine. Paraphyses were filiform, slightly clavate, and varied in size from 62 to 116 by 1 to 3 microns. The dimensions of the spores were 6 to 17 by 2 to 8 microns (averages about 7

to 12 by 4 to 7 microns). The sizes of these bodies fluctuate around the measurements given for *Sclerotinia libertiana*. Ascospore cultures were identical with those isolated from sunflower cankers. These measurements are given because these structures vary in size more than the original description of the fungus indicates.

HISTOLOGY OF SUNFLOWERS WITH SCLEROTINIA WILT

Sclerotinia libertiana rapidly decomposes parenchymatous tissues but does not seem to destroy lignified cells. Large, normal sunflower stems have thick, woody xylem cylinders like a young tree. Sclerotinia mycelium in the medullary rays and cortex disintegrates the unlignified cells, and the lignified tissues often separate into thick strands which give the cankers a shredded appearance (figs. 3, 4). Cross sections of tissues from the base of a rotting sunflower head showed isolated bundles of tracheae and tracheids surrounded by large masses of mycelium in which scattered, disintegrating parenchyma cells occurred; most of the phloem had disappeared.

The mycelium appears to spread most rapidly in the parenchyma cells just below the epidermis, and long hyphae occur abundantly in this region (Pl. II, A). Young cankers appear to involve only the cortical regions at first. Later, the hyphae become common in the medullary rays, phloem, and pith, and are sometimes seen in some of the tracheae (Pl. II, B). The first symptom of infection is the water-soaked appearance of the outer layers of cells. *Sclerotinia libertiana* is one of the parasites which kills its host cells rapidly, with the result that water leaks out of the diseased cells and gives the lesions a water-soaked appearance. Cross sections of Sclerotinia hyphae are shown in Plate II, C and D. These hyphae, and neighboring hyphae seen in lateral view, were 16 to 27 microns in diameter; those in section exhibited dark centers from which irregular protoplasmic strands radiated to the cell walls. Most of the Sclerotinia hyphae seen in sunflower cankers were 7 to 15 microns in diameter.

The vascular bundles are often discolored for 30 to 60 cm. above the external margins of stem cankers. This discoloration is probably due to toxic chemicals conducted up from the cankers, and does not constitute evidence of the presence of myce-

lium far from the cankers. Bisby (5) cultured sunflower tissues taken 7 to 90 cm. above a stem canker and did not secure *Sclerotinia mycelium* in any case. The death and drying of the tissues above the cankers probably stops the spread of the mycelium. In sections of cankered stems hyphae were seen in some tracheae but not in any tracheids or bast cells. Although hyphae occur in the tracheae in old cankers, the fungus does not appear to spread very far through the stem by this means. When cankers occur only in the crowns and bases of stems, there is no evidence of *Sclerotinia* in the seeds borne on these stalks; and healthy, viable seeds probably occur in heads on stalks that show basal infections only late in the season. Many seeds from heads borne on diseased stalks were planted on agar to see whether *Sclerotinia* would develop from them. Since none of the chemicals used for surface disinfection were effective and most of the seeds were quickly overgrown with *Alternaria* and *Penicillium*, definite conclusions can not be drawn from these cultures. However, *Sclerotinia* grows rapidly and well in the presence of these other fungi and, had it been present in the seeds, it should have appeared.

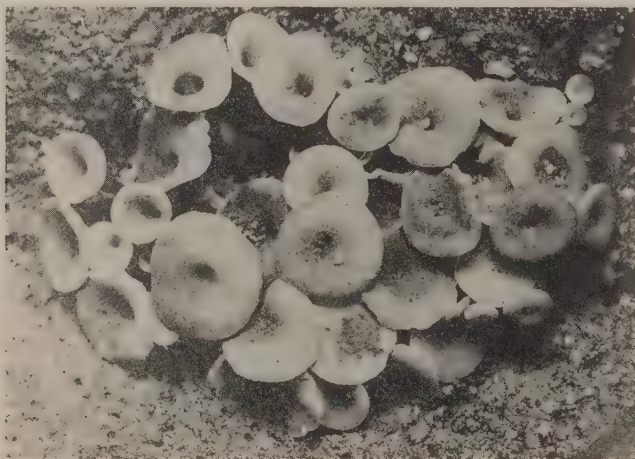


Fig. 5.—Apothecia of *Sclerotinia libertiana* produced by one fragment of a crust sclerotium from a White Beauty sunflower. $\times 11/5$.

DEVELOPMENT OF THE SCLEROTIA OF SCLEROTINIA

Sclerotinia libertiana produces numerous sclerotia of many shapes and sizes. The first stage in the development of a sclerotium under field and laboratory conditions consists of a bunch of white mycelium which rapidly enlarges and becomes rounded; then several droplets of clear, colorless liquid appear on its surface. These droplets vary from 1 to 5 mm. in diameter. The fuzzy, white surface of the sclerotium soon becomes black and nearly smooth, and the water droplets soon run off or evaporate (Pl. III, C). Stevens and Hall (28), Brown (9), and Smith (27) mention the occurrence of these water droplets on developing sclerotia, but no literature explaining the physiology of their production has been seen by the writers.

Sections of young sclerotia before the appearance of the water droplets show a thick crown of tangled hyphae, from the outer margin of which free ends project; there are thick sheaths on some of the hyphae. Boyle (8) gives good illustrations of these hyphal sheaths. There are many wide spaces between the hyphae, and the base of the young sclerotium consists of empty spaces and formless, gelatinous material (Pl. III, A). Soon after this stage is reached, water droplets appear on the surface and rapidly increase in size. Beach (4) illustrates the structure of a young sclerotium of *Sclerotinia minor*.

Sections of smooth, black sclerotia bearing water droplets show that they are very different from the young white ones, for the formless base is absent and the structure of the interior is uniform (Pl. III, B). The interior hyphae are 2 to 4 microns in diameter and all have gelatinous sheaths 2 to 4 microns in diameter which bind them together; a few small, empty spaces between the hyphae were seen in thin sections. The hyphae are granular, guttulate, and appear to contain much stored food. The white interior tissue is completely enclosed by a layer about five cells thick of large, nearly isodiametric cells having thick, brown walls. Sections of old, dry sclerotia did not reveal any constant differences between them and the black sclerotia bearing water droplets. One old sclerotium had brown, partly decomposed, central hyphae which did not stain with acid fuchsin or malachite green, and contrasted sharply with the normal, stained hyphae.

There is a rapid exudation of much water by developing sclerotia. It may be that the physiology of their formation is similar to guttation in cabbage or the exudation of nectar in flowers. The violent spore discharges of *Sclerotinia*, *Pilobolus*, *Empusa*, and some agarics is explained on the basis of hydrostatic pressure. Brown (9) reports oxalate in liquids produced by *Sclerotinia*, and Smith (27) says that the sclerotia and mycelium often become encrusted with calcium oxalate crystals. Van Beyma Thoe Kingma (31) says that oxalic acid occurs in the sclerotial droplets of *Sclerotinia porri*.

We suggest three theories in attempting to explain exudation of water from sclerotia: (1) In the process of maturing, perhaps water is mechanically squeezed out of the sclerotia by the growth pressure of the hyphae which close up the intercellular spaces and develop thick sheaths. (2) Possibly the hyphae of the young sclerotia contain some substance of low osmotic activity which becomes rapidly converted into sugar or some other osmotically active chemical that causes pressures in the hyphae so great that osmotic leakage occurs. The ectoplasmic membranes may filter out part of the solutes. (3) According to a common theory pertaining to the roots of phanerogams, cortical cells adjacent to tracheae have liquids of low osmotic concentration next to the tracheae, and have liquids of high osmotic concentration in these same cells next to the cortical cells that occur between them and the root hairs. This explains osmotic leakage of water from the cortical cells into the tracheae. It may be that this theory of unequal concentrations in cells also explains the exudation of water from sclerotia. The oxalate radical is known to occur in the liquid concerned.

INFECTION BY SCLEROTINIA LIBERTIANA

DeBary (12) placed ascospores in a nutrient solution and found that the resulting mycelium penetrated and killed the tissues of inoculated plants. Since the mycelium from ascospores placed in water failed to cause infections, he thought that the hyphae did not produce chemicals abundantly enough to enable them to penetrate. Stevens and Hall (28) reported that germ tubes of ascospores in water did not penetrate excised lettuce leaves, while mycelium from ascospores in lettuce broth did penetrate similar lettuce leaves. They concluded that

direct penetration by ascospore germ tubes may not occur. Davis (11) saw no infections of living tissues by ascospore germ tubes. Boyle (8) illustrated infections of excised bean leaves by *Sclerotinia* mycelium growing in turnip juice; appressoria produced penetrating hyphae which entered the leaves. Ramsey (25) says that this fungus can penetrate without wounds.

Although vigorous *Sclerotinia* mycelium can penetrate leaves, most of the available evidence indicates that it usually enters its hosts through wounds. Branch roots normally originate in the pericycle and cause innumerable small wounds when they rupture their way out through the cortical and other tissues. Movement of rigid stems by the wind breaks small roots; cultivation, hail, and birds cause other wounds in sunflowers. Hence, wounds are sufficiently numerous to permit rapid spread of *Sclerotinia* in the field and, as shown in the section on spread of wilt, *Sclerotinia* spreads rapidly when the sunflowers are not more than 30 cm. apart. Data on infection by *Sclerotinia* are given in the section on inoculation experiments.

Weaver (34) showed that the lateral roots of sunflowers spread out from 0.6 to 1.6 m. from the stalks, and that numerous small roots occupy the upper 0.6 m. of the soil. The intermingling of the branch roots of sunflowers in the same and adjacent rows exposes many plants to infection by the *Sclerotinia* mycelium in scattered, diseased plants, so that it is probable that many branch roots become infected. The appearance of many crown rots in August and September may be partly explained on the basis of earlier infections that finally reached the crowns through branch roots 1 to 2 m. long; probably many of these infections never reach the crowns and produce aerial symptoms. Since it is not evident that *Sclerotinia* spreads between rows 1 m. apart, and spread is not serious between plants more than 30 cm. apart in the rows, it is tentatively concluded from this circumstantial evidence that spread of the fungus through long branch roots is slow.

When *Sclerotinia* began to attack a large number of heads in the sunflower plot in 1925, the means of infection of heads high above the ground was not understood. *Sclerotinia libertiana* produced no spores during the dry summer weather here, and there was no evidence that the fungus grew very far from

its cankers in the stems. An explanation of the means of infection of the heads became apparent with continued observation. From the time that the sunflower seeds began to ripen, the plot and neighboring fields were visited daily by flocks of birds. Since the White Beauty sunflowers in the plot ripened earlier than the Mammoth Russian sunflowers in the fields, birds visited the plot in large numbers, and it was necessary to tie sacks over all the heads desired for seed.

The birds were very tame and their feeding was often observed within distances as short as 2 or 3 m. Most of the sunflower heads faced the ground, so the birds hung on to the margins of the heads with their claws and reached under the heads to secure the seeds, thus making many small wounds in the margins of all the heads in the plot. With dexterous twists of their bills, they cracked off the hulls of the seeds and caught the embryos. The birds succeeded in catching the embryos only part of the time, so that they were constantly flying to the ground to secure the food they had missed. As shown by the rapid spread of wilt in this plot, the soil was infested with *Sclerotinia*. The birds used a rather effective method of inoculation in transferring mycelium from diseased heads and the soil to the wounds which they scratched in the backs of healthy heads. This probably explains the appearance of *Sclerotinia* rots in 113 heads on erect stalks in this plot in 1925. Many heads rotted in the adjacent field of Mammoth Russian sunflowers. Early freezes killed the sunflowers in 1926 before any seed ripened, and no head rots were seen. A small number of heads were rotted by *Sclerotinia* in 1927.

HOST RANGE OF *SCLEROTINIA LIBERTIANA*³

Jones (16) gives European records of the occurrence of *Sclerotinia* in *Helianthus*. Saccardo, in the "Sylloge Fungorum," Stevens and Hall (28), Stevens (29), Bisby (5), Rees (26), Massee (19, 20), and the present writers report that this fungus causes diseases in the following plants:

³Names marked with an asterisk represent hosts in which this fungus is not known to have been previously reported.

Aegopodium	*Fanweed (<i>Thlaspi</i>)	Onion
Artichoke	Fennel	Parsley
Bean	*Field mustard	Pea
Beet	(<i>Brassica</i>)	Petunia
Cabbage	Forsythia	*Pigweed
Campanula	*French endive	(<i>Amaranthus</i>)
Canada thistle	(<i>Cichorium</i>)	Potato
Caraway	Ginseng	Radish
Carrot	Hemp	Rape
Cauliflower	Hollyhock	*Rutabaga
Celery	*Lamb's quarters	Snapdragon
Cerifolium	(<i>Chenopodium</i>)	Sow thistle
Chicory	Lemon	(<i>Sonchus</i>)
Chrysanthemum	Lettuce	*Sugar Beet
Clover	Marsh elder (<i>Iva</i>)	Tomato
Columbine	Mulberry	Vetch
Cucumber	Mustard	Wild sunflower
Daucus	Omphalodes	(<i>Helianthus annuus</i>)
		Zinnia

Sclerotinia libertiana killed many plants of *Brassica arvensis* (L.) Ktze, that grew near wilting sunflowers. Nine of the mustard stems contained sclerotia in their pith cavities, and three tap roots bore dense, white mycelium. The crown cankers were like those in the sunflowers; a few mustard cankers had a shredded appearance.

Many fanweed plants (*Thlaspi arvense* L.) near wilting sunflowers were killed and bore basal cankers like those of sunflower wilt. Six pigweeds (*Amaranthus retroflexus* L.) growing close to wilting sunflowers in the experimental plot in 1925 were killed by *Sclerotinia*. One pigweed tap root bore dense, white mycelium and the pith cavity in a stem contained a sclerotium; the stem and root cankers resembled sunflower cankers. A plant of *Chenopodium album* L. near a wilting sunflower bore a basal canker 10 cm. long which was like the one in the sunflower.

Canada thistles (*Carduus arvensis* (L.) Robs.) were commonly killed by *Sclerotinia libertiana* when they grew close to wilting sunflowers. This fungus was isolated several times from wilting thistles. The underground stems were killed and became brown, and sclerotia were seen often in the cavities decayed by the fungus in these thistle stems. In one commercial sunflower field, wilt seemed to be most severe in a patch

of Canada thistles. The weeds considered here appeared to be attacked by *Sclerotinia* only when they were growing near wilting sunflowers.

In 1925 some wilting sunflowers were transplanted into a plot of hemp (*Cannabis sativa* L.) Ten weeks later, four of the hemp plants near the sunflower stalks had developed root and basal stem cankers that resembled those in the sunflowers. The hemp roots probably had been killed by *Sclerotinia*, for they bore white mycelium and sclerotia. A dense, white mold was found on some French endive (*Cichorium*) roots growing



Fig. 6.—A White Beauty sunflower head decomposed by *Sclerotinia* (field infection) showing the remaining fibrous tissues, seeds, and sclerotia. $\times 11/18$.

15 m. from the sunflower plot. Cultures on agar and on these roots in moist chambers showed that the fungus was *S. libertiana*. In inoculation experiments rutabagas and sugar beets were rotted by this fungus.

MEANS OF LIVING THROUGH THE WINTER

Sclerotia and mycelium of *Sclerotinia libertiana* live over winter in the field on and in diseased sunflower stalks and in the soil; this fungus appears to be well adapted to saprophytic growth. Seeds from diseased sunflower heads often have small sclerotia attached to them. Two cases of sclerotia inside seed coats were seen, so sclerotia in seed coats may be rather common (Pl. III, D). If diseased heads are mixed with the normal heads in harvesting and threshing sunflower seeds, the whole lot of seed would be contaminated with fragments of sclerotia

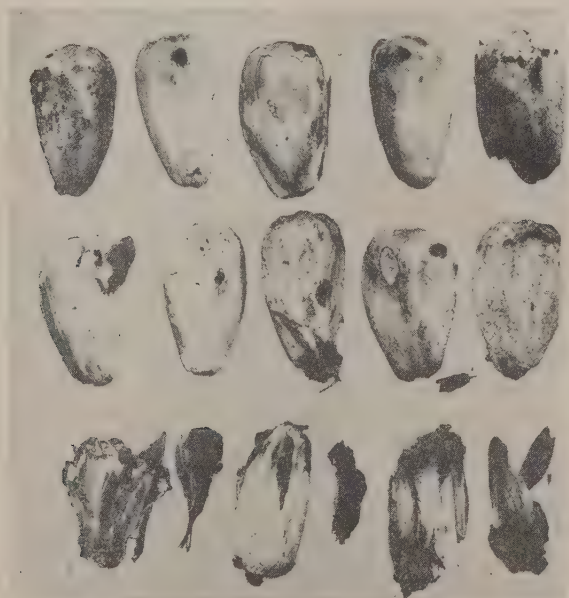


Fig. 7.—White Beauty sunflower seeds from a head rotted by *Sclerotinia*, showing sclerotia attached to the seed coats and chaff. $\times 9/5$.

and seeds with sclerotia attached to them, thus making seed dissemination of *Sclerotinia* probable.

Three samples of sclerotia brought in from the field and cultured on February 17, 1926, produced mycelium and new sclerotia. Three of the sclerotia brought in from the field on April 28, 1923, produced apothecia when placed on agar. Mycelium which had overwintered on sunflower stalks in the field was brought in and placed on agar on March 30, 1923; all the plates developed *Sclerotinia*. Bisby (5) and Baribeau (3) described the means by which *Sclerotinia* lives through the winter.

INOCULATION EXPERIMENTS

Extensive inoculation experiments by the authors, and by Bisby (5), Lawrence (18), and Jones (16) have proved that *Sclerotinia libertiana* is the cause of sunflower wilt. In 1925 this fungus was isolated from a wilting Mammoth Russian sunflower, and transfers of one of the cultures were used in the subsequent inoculation experiments.

The inoculations considered in Table I were made in 1925 in the White Beauty sunflower plot; only normal, healthy tissues were inoculated. The inoculations were made at short intervals throughout the summer, so that the sunflowers were from 0.3 to 2.3 m. tall when they were inoculated. Most of the infections that occurred at all appeared within 3 to 14 days after the inoculation of the plants. Infections occurring in inoculated plants later than three weeks after inoculation were called natural infections. The percentages of infections were high early in the season and low late in the season because the old tissues were less susceptible than the young ones. While 427 plants were artificially inoculated, 4,442 other plants in this plot were not artificially inoculated; during the summer 27.3 per cent of these latter plants became naturally infected. Scattered through this plot were 218 sunflowers which had become infected through artificial inoculations, and it was obvious that *Sclerotinia* had spread naturally from them to neighboring healthy plants in many cases.

The fungus was reisolated from a stem canker caused by an artificial inoculation, and was found to have the same characteristics as laboratory cultures of *S. libertiana*. On August 27, 1925, it was reinoculated into four large, healthy sunflower

stalks in which it caused four typical cankers 7 to 30 cm. long. Some of the newly diseased bark on these plants was pink in color, and one canker had a brown border.

In 1920 many sunflowers were killed as a result of artificial inoculations with *S. libertiana* while many check plants remained healthy. In greenhouse experiments in 1926, 23 wound inoculations of White Beauty and Mammoth Russian sunflowers 0.5 to 1.5 m. tall resulted in 20 typical cankers within five days. Seven of these infections were head rots, 5 were root rots, and 8 were stem rots. Uninoculated plants in the pots with these diseased plants were all killed within six weeks. Eight similar sunflowers in neighboring pots were not inoculated and served as checks; all remained healthy.

TABLE I.—FIELD INOCULATIONS OF WHITE BEAUTY SUNFLOWERS WITH *SCLEROTINIA LIBERTIANA* IN 1925. ONLY THOSE INFECTIONS WHICH OCCURRED WITHIN 3 TO 21 DAYS WERE COUNTED.

Method of Inoculation	No. of in- oculations	Number successful	Percent successful
Moldy tap roots of wilting plants placed near wounds in tap roots of healthy plants.....	99	62	62.6
Moldy tap roots of wilting plants placed near wounds in branch roots of healthy plants.....	57	44	77.4
Sclerotinia mycelium on agar placed in shallow wounds in healthy tap roots	89	43	48
Check—Tap roots wounded, but not inoculated.....	2	0	0
Wounds made in tap roots of healthy plants growing near wilting plants	4	2	50
Sclerotinia mycelium on agar placed in shallow stem wounds and covered with wet cotton.....	58	26	45.4
Sclerotinia mycelium on agar placed in deep stem wounds and covered with cotton.....	58	30	52
Checks—Healthy stems wounded, but not inoculated, and wounds covered with wet cotton.....	10	0	0
Sclerotinia mycelium on agar placed on unwounded stems and covered with cotton.....	5	1	20
Sclerotinia mycelium on agar placed in deep stem wounds, but not covered with cotton (late summer)	14	0	0
Sclerotinia mycelium on agar placed in deep wounds in tops of broken stalks and covered..	10	0	0
Sclerotinia mycelium on agar placed on young, unwounded disc flowers and covered with wet cotton	2	2	100
Sclerotinia mycelium on agar placed in deep wounds in the backs of old, green heads and covered with wet cotton	20	8	40
Mycelium on agar placed in deep wounds in backs of heads, but not covered (in late summer)	11	0	0
Totals and percentage of successful inoculations	427	218	51

In 1920 lettuce plants inoculated with *Sclerotinia* from sunflowers developed typical symptoms and signs of lettuce drop as described by Stevens and Hall (28). This fungus rotted carrots, rutabagas, and sugar beets inoculated with it.

In the White Beauty sunflower plot in 1926, 29 sunflowers 30 to 60 cm. tall were inoculated with an ascospore culture of *S. libertiana* by placing the mycelium on agar in shallow stem wounds and covering the wounds with wet cotton. Within five days 14 of the inoculations had resulted in typical stem cankers, and 4 other cankers appeared within the next ten days; thus 62 per cent of these inoculations were successful. More than 100 similar, uninoculated plants in this plot remained healthy all summer. Five inoculations of one White Beauty and two Mammoth Russian sunflowers with an ascospore culture in the greenhouse all resulted in typical root and stem cankers within three to seven days; two check plants remained healthy long after the inoculated plants had died.

Detailed experiments were performed to determine whether *Sclerotinia* would penetrate the uninjured stems of sunflower seedlings. Treatment of a large number of unhulled sunflower seeds with Semesan, Upsulun, and mercuric chloride failed to result in aseptic seedlings, so it was necessary to hull some old Mammoth Russian sunflower seeds and treat the embryos with 0.1 per cent mercuric chloride solution, followed by three washings with sterile distilled water. The treated embryos were transferred aseptically to the wet filter paper in the bottoms of large, autoclaved dishes of the Petri dish type; all treated seeds were handled in this manner. The dishes were kept in a dark room at 20°C. for five days until the plumules were 2 to 6 cm. long. Aseptic seedlings resulted from the treated embryos, so they were inoculated by placing mycelium of *Sclerotinia libertiana* on agar on the stems. The seedlings were then rolled in wet, autoclaved rags and placed in large, sterile test tubes.

Within a week after inoculation the seedlings were examined by inserting the point of a sterile needle under the outer 1 to 3 layers of cells of the inoculated regions and pulling off thin strips of tissues, which were then stained with acid fuchsin and mounted in glycerine. *Sclerotinia* did not penetrate the uninjured stems of any of the seedlings examined. Since these

inoculations were tried in only two series, and because *Sclerotinia* did not penetrate some seedlings that were wounded with the point of a sterile needle before inoculation, the results of these penetration studies are not conclusive. However, the methods used here have been successful with other fungi many times, so these results support the general opinion that *S. libertiana* usually enters its hosts only through wounds.

SPREAD OF SCLEROTINIA LIBERTIANA IN SUNFLOWER FIELDS

This fungus usually enters its hosts through wounds. Wounds are commonly present in such numbers in field sunflowers that *Sclerotinia* has plenty of infection points for rapid spread. Wind movement of the rigid stems breaks small roots, and the development of branch roots causes many small wounds. Hail, cultivation, and birds cause other wounds. Spread of this soil-inhabiting fungus in the rows is very rapid in the field when the stalks are not at least 30 cm. apart.

TABLE II.—PERCENTAGES OF WILTED SUNFLOWERS IN THE MAMMOTH RUSSIAN SUNFLOWER PLOTS OF THE AGRONOMY DEPARTMENT AT BOZEMAN ON AUG. 25, 1921, SHOWING THE EFFECTS OF DISTANCE OF PLANTING UPON THE SPREAD OF WILT.

	Rows 60 cm. apart				Rows 1 m. apart			
	10 cm.	10 cm.	20 cm.	30 cm.	10 cm.	10 cm.	20 cm.	30 cm.
Distance between stalks.....	10 cm.	10 cm.	20 cm.	30 cm.	10 cm.	10 cm.	20 cm.	30 cm.
Number of stalks counted.....	523	658	235	190	525	547	265	180
Number wilted	180	184	28	9	212	249	32	6
Per cent wilted	34.4	28	11.9	4.7	40.4	45.5	12.1	3.3

The data presented in Table II show that the rapidity of the spread of *Sclerotinia* in the rows was decreased by thinning the stalks until they were at least 20 cm. apart. In commercial fields the stalks in the rows are usually about 10 cm. apart.

In the White Beauty sunflower plot in 1925, 148 successful root inoculations were made during the season. The resulting diseased stalks served as centers from which *Sclerotinia* spread to the healthy stalks. This plot was not cultivated after the middle of June. The following figures give the number of uninoculated plants in this plot which showed natural infection with *Sclerotinia* on the different dates:

June 29th	None of the 4,869 plants showed infections
July 27th	143 showed wilting or crown rots
August 7th	258 showed wilting or crown rots
September 3rd	737 showed wilting or crown rots
September 14th	837 showed wilting, crown rots, or head rots
October 3rd	1174 showed wilting, crown rots, or head rots
October 21st	1210 showed wilting, crown rots, or head rots
On October 21st (including 218 artificially inoculated plants), there was a total of 1,428, or 29.3 per cent, of the sunflowers in the plot diseased with Sclerotinia.	

In this plot, thinning the stalks until they were more than 30 cm. apart decreased the spread of wilt as it did in the plots concerned in Table II.

In rows 1 to 10, there were 1050 plants 15 to 20 cm. apart, of which 177 wilted.

In rows 11 to 23, there were 1472 plants 30 cm. apart, of which 205 wilted.

In rows 24 to 33, there were 1780 plants 8 to 15 cm. apart, of which 738 wilted.

In rows 34 to 42, there were 425 plants 40 to 50 cm. apart, of which 75 wilted.

In rows 43 to 46, there were 142 plants 45 to 90 cm. apart, of which 15 wilted.

In 1926, 10 rows of White Beauty sunflowers were planted in the positions occupied by rows 35 to 44 in 1925. This plot was cultivated once about July 25th, and at no other time during the growing season. None of the plants were artificially inoculated. By September 26th 32 of the plants had wilted, most of them growing on the ground occupied by rows 35 to 41 in 1925. The first row, which contained 155 stalks about 15 cm. apart, showed 9 per cent wilt. The stalks in the other rows were 25 to 50 cm. apart and occurred on soil less heavily infested with Sclerotinia. In 1925, 104, or 23 per cent, of the sunflowers on this ground wilted, but 25 of these stalks wilted from artificial infections early in the season. The decrease in wilt in 1926 is explained on the basis of no artificial inoculations, wide spacing of the stalks, and an apparently less favorable season. Sunflower wilt had never been serious on this ground before 1925. Sunflower wilt was a little less severe in the Agronomy Department plots in 1926 than in 1925, as shown in Tables III and IV.

TABLE III.—PERCENTAGES OF WILTED SUNFLOWERS IN THE MAMMOTH RUSSIAN SUNFLOWER PLOT OF THE AGRONOMY DEPARTMENT AT BOZEMAN IN 1925.

Plot Number	July 23rd		Aug. 6th		Aug. 25th		Sept. 16th
1	*15.0%	30.0%	34.5%	24.0%	21.0%	37.5%	54
2	19.5	10.5	33.5	24.5	33.0	36.0	72
3	20.5	24.0	35.5	23.5	56.5	64.0	85
4	25.5	32.5	21.5	51.5	53.0	57.0	92
5	37.0	22.5	51.0	48.0	52.5	52.5	81
6	29.0	25.5	50.5	46.5	61.0	49.5	74
7	23.0	16.0	44.0	24.5	50.0	29.5	54
Averages	24.3	23.0	39.0	35.0	47.0	47.0	73

*The double percentages given for the first three dates indicate the variations that occurred in the plots.

TABLE IV.—PERCENTAGES OF WILTED SUNFLOWERS IN THE MAMMOTH RUSSIAN SUNFLOWER PLOTS OF THE AGRONOMY DEPARTMENT AT BOZEMAN IN 1926.

Plot No.	No. plants in plot	July 17th	July 23rd	July 31st	Aug. 14th	Sept. 1st	Sept. 13th
2	2236	2.0%	6.4%	14.4%	29.3%	44.0%	42.4%
3	1742	3.6	10.8	25.0	49.0	72.2	77.1
4	2067	4.4	10.3	23.1	42.4	56.5	53.0
5	1950	5.0	13.0	30.0	51.5	66.8	62.0
6	2197	3.0	6.9	16.5	30.3	44.2	42.4
7	2015	3.0	5.8	13.9	28.2	37.4	42.1
Averages		3.5	8.9	20.5	38.5	53.5	53.1

The plots considered in Tables III and IV have been planted in sunflowers continuously since 1920. In 1925 there were about 2,000 plants in each plot; the percentages in the first two plots should be higher because more than 125 wilted plants were removed before the counts in August were made. The plants in the rows were about 10 cm. apart. In 1925 each percentage is based on a count of 200 plants in the order that they occurred in the rows.

In 1926 all the diseased plants in all the plots were counted on each of the first five dates; on the last date the wilted plants in three representative rows were counted and the percentages computed from these figures (Table IV). Identical plots are indicated by the plot numbers in Tables III and IV. The yields in these plots in 1925 and 1926 were 7.2 to 15.4 tons per acre (average of 10.6 tons). These yields contrast sharply with the five-year average yields of these plots from

1920 to 1924, which were 20.8 to 23.4 tons per acre (average of 21.7 tons). Wilt was the chief factor in this 50 per cent reduction in the yields; the effects of continuous cropping was the other main factor. These data on yields were furnished by the Agronomy Department of the Experiment Station. On September 6, 1927, when they were cut, 20 to 50 per cent of the sunflower plants had wilted in five plots on this ground. Wilt was spreading rapidly.

In 1920, 1921, 1925, 1926, and 1927 thousands of sunflower plants in commercial fields were counted to determine the percentages of wilted plants. This disease was not observed in the Flathead Valley, while fields in Ravalli County in 1920 showed traces to 7 per cent of the sunflowers wilted. In this county in 1925 and 1926 sunflower fields showed traces to 80 per cent of the plants wilted. These observations showed that the disease increased in severity during this period of eight years.

Sunflower wilt appears to be a cool-weather disease. In North America it has been reported only in the northern states and Canada. The following records of the soil temperatures in the experimental plots at Bozeman have some significance. During the period of most rapid spread of *Sclerotinia* in 1925, which was from July 1 to August 7, the temperatures of the upper 15 cm. of soil in the sunflower plot varied from 3 to 25°C., with averages around 9 to 18°C. From June 28 to September 20, 1926, the temperatures of the upper 15 cm. of soil near the sunflower plot varied from 6 to 24°C., with averages around 10 to 20°C. The air temperatures during these two growing seasons varied from 0 to 32°C., with averages around 10 to 25°C. These records show that the destructive wilting caused by *Sclerotinia* occurred while air and soil temperatures did not average above 20°C.

CONTROL OF SUNFLOWER WILT

Sclerotinia libertiana is a strong facultative parasite which lives in the soil and usually attacks sunflowers through their roots. Like other facultative root parasites, it can be spread in infested soil by irrigation and drainage water, and in mud and dirt on agricultural implements and the feet of animals. This fungus has a very wide host range that particularly includes

garden plants and root crops. Seeds from diseased heads often bear sclerotia attached to them and fragments of sclerotia are mixed with them. No high percentages of sunflower wilt were seen in fields where rotation was practiced. This knowledge makes it possible to make the following recommendations for the control of *Sclerotinia* sunflower wilt: (1) Use at least a four-year rotation which includes cereals and other grass crops, for these crops appear to be practically immune to *S. libertiana*. Leguminous hay crops are not liable to be attacked seriously. (2) Plant good seed which is normal in color and free from sclerotia. In the experimental plots thinning the stalks in the rows until they were 0.6 to 1 m. apart gave good control of sunflower wilt because it delayed the spread of *Sclerotinia* from diseased to healthy plants. This practice is not recommended because it results in a low yield of coarse stalks.

SUMMARY

Sunflower wilt was discovered in Montana in 1920, and investigation of the disease was started immediately.

Sclerotinia libertiana causes a wilt of cultivated sunflowers which kills from 0.1 to 80 per cent of the plants in commercial fields, and often causes serious damage when rotation is not practiced. A loss of 0.1 to 10 per cent is common in fields where rotation is practiced.

The main symptoms and signs of sunflower wilt are the sudden wilting of the leaves and the drooping of the tops, the decayed roots, the brown stem and crown cankers which often bear external and internal mycelium and sclerotia, the shredded and broken cankers, the rotted heads, and the presence of sclerotia on the heads and roots.

Mycelium of *S. libertiana* rapidly decomposes parenchymatous tissues, but does not seem to attack lignified tissues. Hence the lignified strands often separate and give the cankers a shredded appearance.

Sclerotinia hyphae occur in the tracheae in old cankers, but they do not grow in them very far beyond the canker margins.

In 1927 at least 1,000 apothecia of *Sclerotinia libertiana* were produced in a field by sclerotia left in the soil by wilted plants in the preceding crop.

Crust sclerotia as large as 12 by 20 by 0.5 cm. were seen in sunflower heads.

Large sclerotia occurred in the pith cavities in rotting heads; others occurred on and in seeds, and surrounding roots.

S. libertiana is a wound parasite. Wounds in sunflowers in the field are caused by cultivation, hail, birds, movements of the rigid stems due to wind, and by the development of branch roots.

S. libertiana spreads very rapidly in rows of sunflowers unless the stalks are at least 30 cm. apart.

In securing seeds, birds appear to inoculate sunflower heads with *Sclerotinia* by carrying mycelium from diseased heads and the soil to wounds which they scratch in the healthy heads.

S. libertiana has a very wide host range. *Brassica arvensis*, *Thlaspi arvense*, *Amaranthus retroflexus*, *Chenopodium album*, French endive (*Cichorium*), rutabagas and sugar beets are added to the list of known hosts.

Ascospore culture of *S. libertiana* caused typical cankers when they were inoculated into sunflowers in the greenhouse and in the field.

S. libertiana killed 51 per cent of the 427 sunflowers inoculated with it in the field in 1925. It also killed many sunflowers inoculated with it in the greenhouse.

Methods recommended for the control of sunflower wilt are a four-year rotation with cereal and hay crops, and use of clean seed, and agricultural sanitation.

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EXPLANATION OF PLATES

Plate I

A & B. Two black sclerotia of *Sclerotinia libertiana* with some white mycelium attached, from cavities decayed in White Beauty sunflower stems. The sclerotium photographed in B was 118 mm. long.

C. Part of a flat sclerotium of *S. libertiana* borne on the receptacle of a White Beauty sunflower head just below the seed layer. Part of the black surface is covered with white mycelium. This fragment, when photographed, was 44 mm. long.

D-G. Sclerotia of *Sclerotinia libertiana* borne in the spongy tissues below the seed layers in White Beauty sunflower heads. The longest dimension of F, when photographed, was 87 mm. and that of E was 45 mm.

H, L, M, N. Sclerotia of *S. libertiana* surrounding branch roots of Mammoth Russian sunflowers. When photographed, L was 23 mm. long and contained a central, longitudinal hole from which a root was removed.

I-K. Crust sclerotia of *S. libertiana* borne in the seed layers of White Beauty sunflower heads in 1925. Holes occur in the sclerotia which formed around the sunflower seeds; in J, the ends of three seeds are seen in the holes. K shows the lower surface of a sclerotial crust. When photographed, I was 85 mm. long.

Plate II

A. Photomicrograph of a longitudinal section of a cankered White Beauty sunflower stem showing long hyphae of *Sclerotinia libertiana* in the parenchyma cells below the epidermis. $\times 168$.

B. Photomicrograph of a longitudinal section of a White Beauty sunflower stem showing hyphae of *S. libertiana* in and near the tracheae. $\times 130$.

C & D. Photomicrographs of a cross section of a White Beauty sunflower stem showing cross sections of the hyphae of *Sclerotinia libertiana* in the phloem and parenchyma cells. Many of these hyphae have dark centers from which irregular strands of protoplasm radiate to the cell walls. The bundle shown at the top of D is the one shown in C. A normal bundle with adjacent bast is shown at the bottom of D. C is magnified 170 times and D, 62 times.

Plate III

A. Photomicrograph of a section of a young sclerotium showing the prominent sheaths on the hyphae, the large intercellular spaces, and an early stage in the formation of the exterior crust. This fuzzy, white sclerotium of *Sclerotinia libertiana* was imbedded before the water droplets appeared. $\times 270$.

B. Photomicrograph of a section of an old, dry sclerotium of *Sclerotinia libertiana* showing the thick exterior layer of brown cells, and the white interior composed of tightly packed, granular, guttulate hyphae cemented together by their sheaths. $\times 313$.

C. *Sclerotinia libertiana* developing in pure culture on an autoclaved sunflower stem showing the numerous, large water droplets on the young, black sclerotia. $\times 2$.

D. A sclerotium of *S. libertiana* and part of the White Beauty sunflower seed coat inside which it developed; it grew in a badly rotted head. $\times 2$.

E. Apothecia of *S. libertiana* all produced by one fragment of a crust sclerotium from the seed layer of a White Beauty sunflower. The sclerotium is partly visible. $\times 12/5$.

